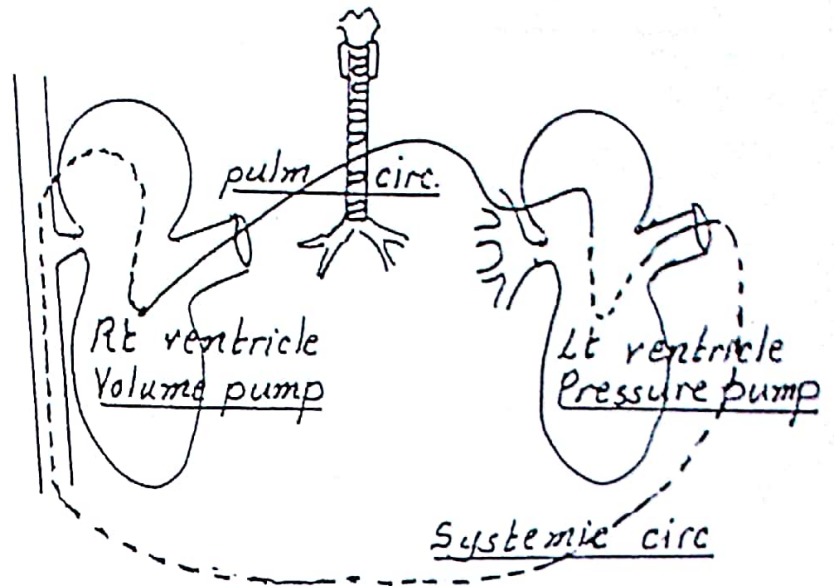


Cardio vascular system

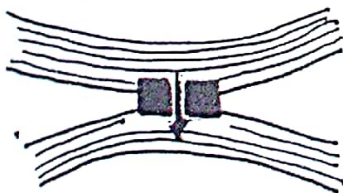
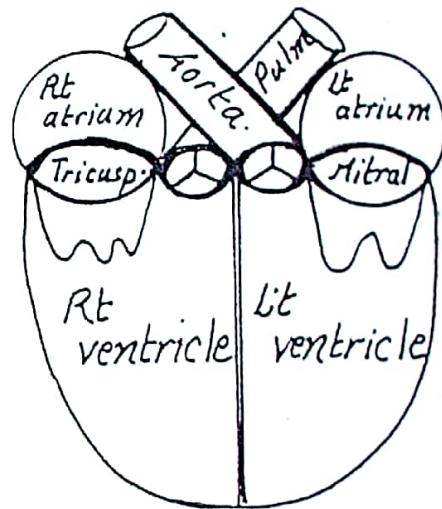
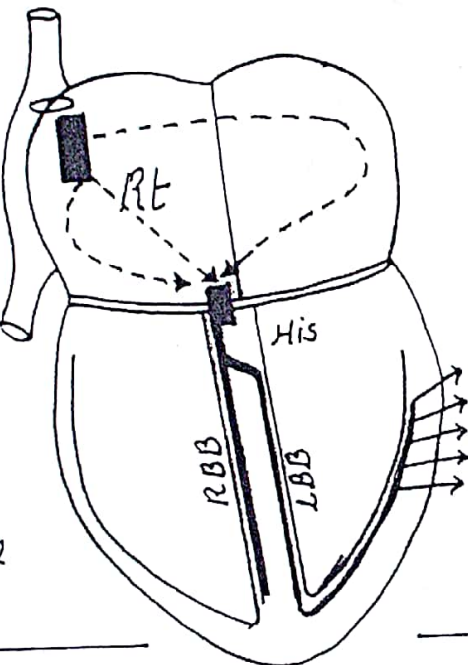
- Two pumps
- Two chambers
- Two circulations
- Two valves
- Two fibres



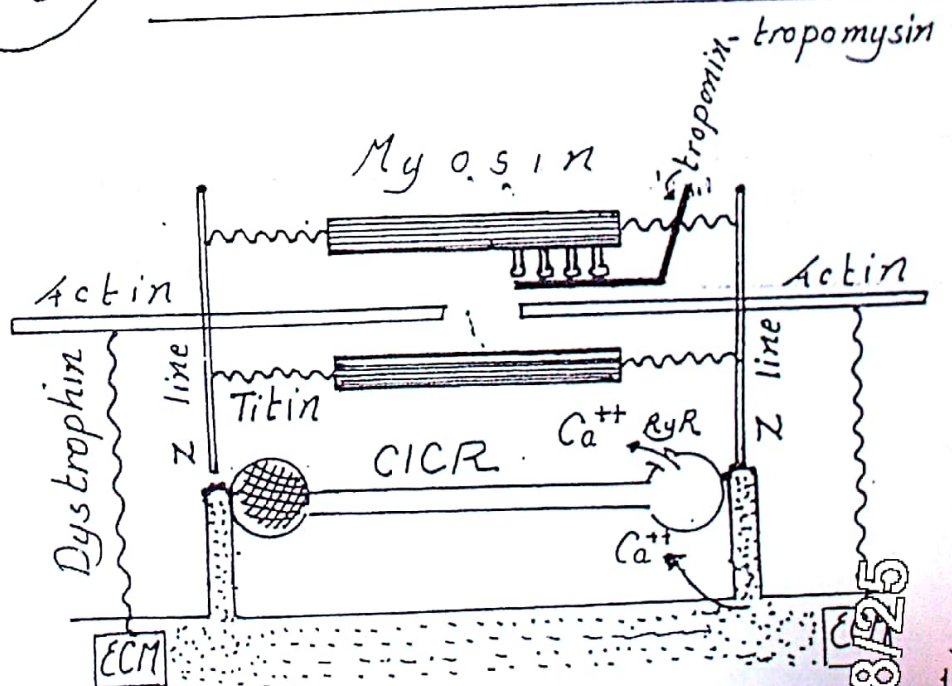
SA node
90/Min

AV node
60/Min

Purkinje
system
30/Min



Intercalated disc
Gap junction



VISIT...

LANZAROTE
Caliente.COM

1 Excitability and electrical properties

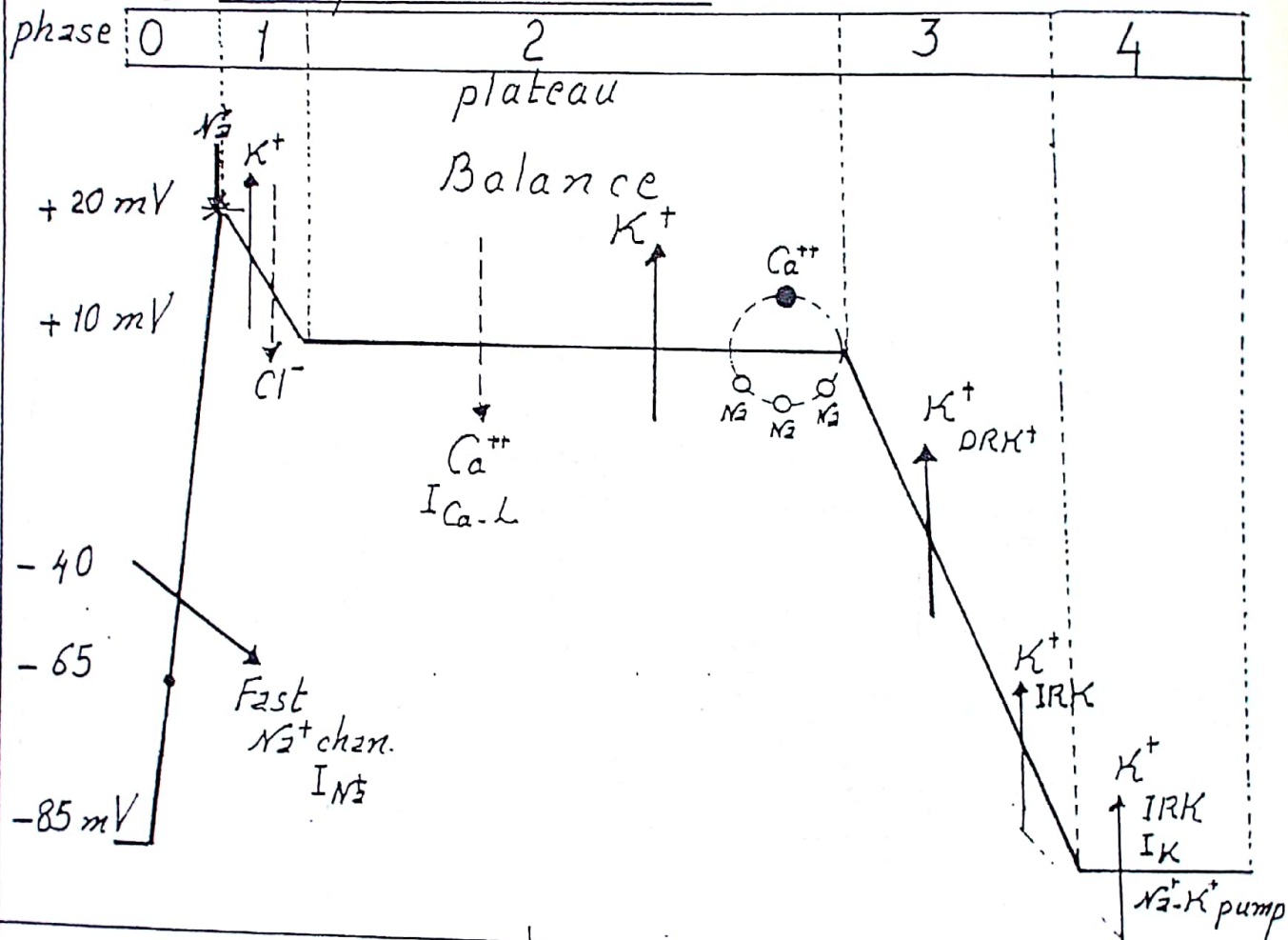
• RMP - 85 mV

• Monophasic action potential

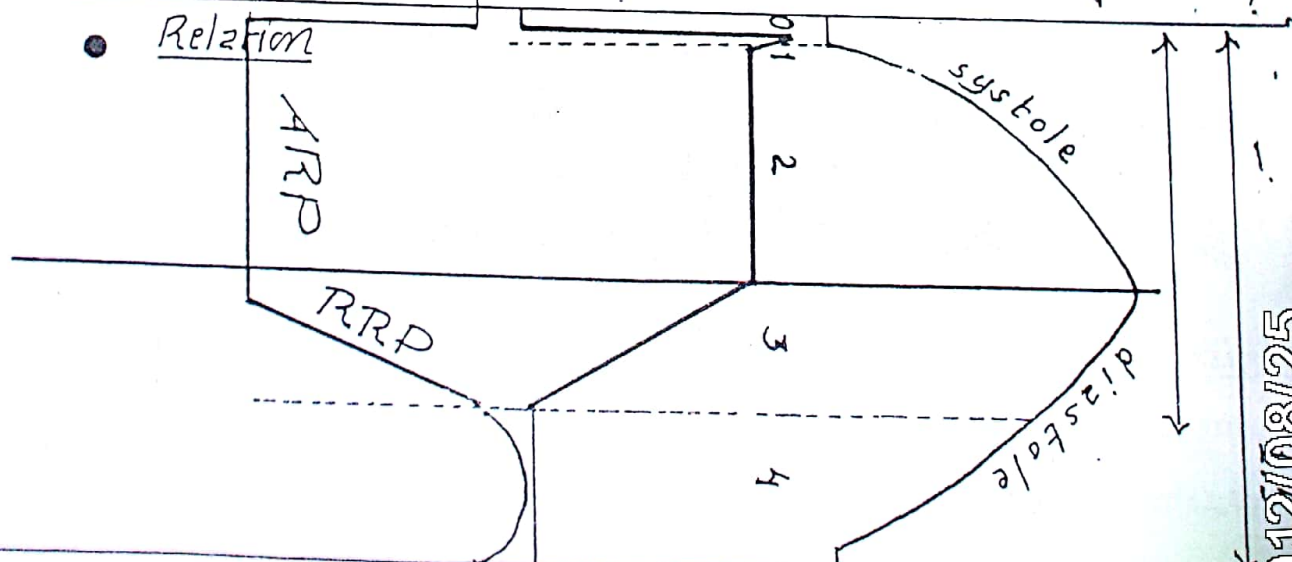
• Recording

• Duration Long Atrial 200 Vent 300 msec.

• Description & ionic bases

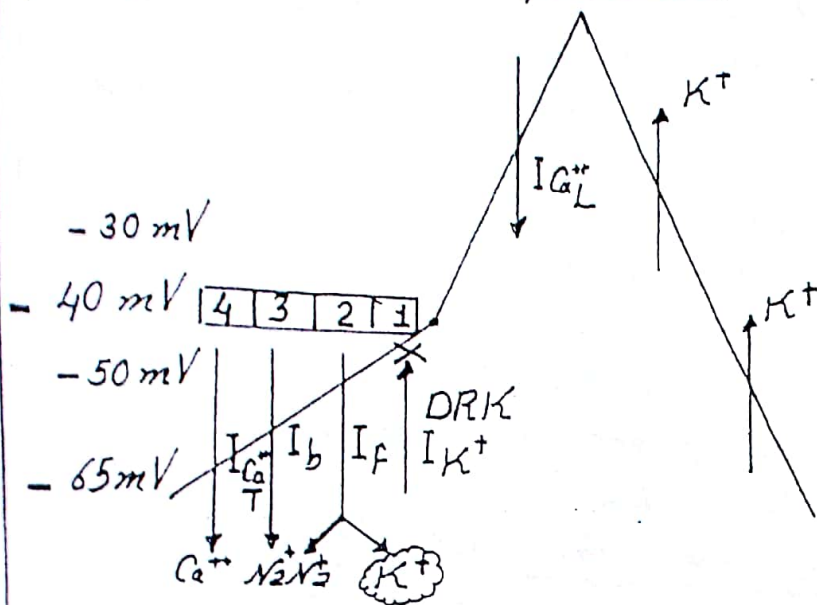


• Relation



2 Rhythmicity (Automaticity)

+10 mV Nodal action potential



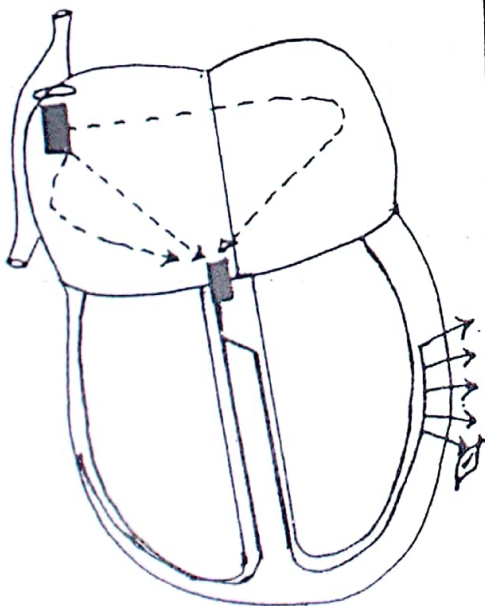
No stable RMP . No plateau
spontaneous slow diastolic depol
slow upstroke
Apex of A. pot +10 mV

SA node pace maker
Highest rhythm

Control

- 1 Vagus Acetylcholine
M₂ receptors
++ K⁺ conduct.
Hyperpol.
90 -- HR → 70/min
- 2 Symp Norepinephrine
β₁ ++ cAMP
open L Ca²⁺ ch.
Depol
++ HR →
- 3 Temp (fever) ++ HR
- 4 Digitalis -- HR

3 Conductivity Spread of cardiac excitation



<u>Tissue</u>	<u>Velocity m/s</u>	<u>Time in sec.</u>
• SA node	0.05	
• Internodal tract (atrial)	1	0.1
• AV node	0.05	0.1 <u>SLOWEST</u>
• Bundle of His	1	
• Purkinje system	4	<u>FASTEST</u>
• Ventricular ms	1	0.1

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4 Contractility

● Excitation - Contraction coupling:

Contraction $\xrightarrow{\text{Na current}}$ Ca^{++} influx via $\text{I}_{\text{Ca}^{++}\text{L}}$
 $\xrightarrow{\text{Ca}^{++}}$ Ca^{++} release from SR via RyR i.e. CICR

Relaxation caused by Ca^{++} via 3 mechanisms

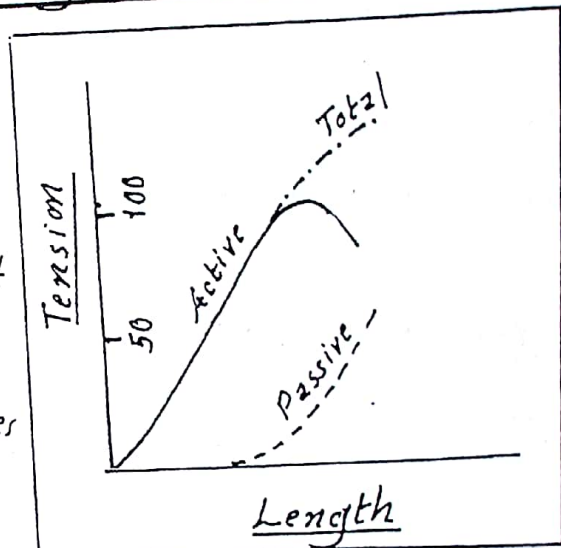
- 1 Ca^{++} pump into SR
- 2 Na^{+} - Ca^{++} exchanger
- 3 Calmodulin

N.B Ca^{++} release process is recovered by Na^{+} - K^{+} pump
 Activation of Cardiac ms by CICR is BELOW maximum

● Correlation between m. Fibre length and tension:

Progressive ++ in length
 via preloads:

- 1 $\rightarrow$ ++ passive tension without stimulation
 - 2 $\rightarrow$ ++ active tension within limit with stimulation i.e. Starling law
- Mech ++ number of cross bridges
 ++ affinity of Ca^{++} to troponin C



N.B C. muscle contracts isometrically then isotonicly
 i.e. Tension 1st ++ then constant
Afterload the tension at which the load is lifted.

● Performance of C muscle (isolated)

is affected by:

- 1 Preload resting length
- 2 Afterload
- 3 Inotropic state
- 4 Frequency of contraction

is measured by:

- 1 Extent of shortening ΔL
- 2 Velocity of shortening dL/dt
isotonic cont.
- 3 Active tension
isometric cont

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Effect of preload and afterload on

1 Length tension curve

Length shortening curve

Afterload

Preload

⊕⊕ within limits

At the same level of

Progressive ⊕⊕ in

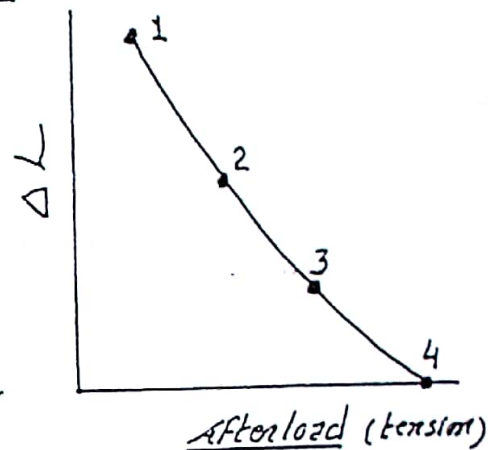
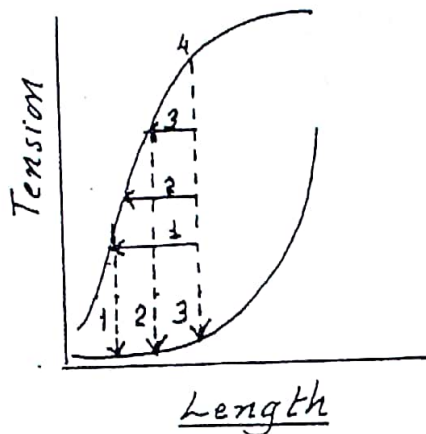
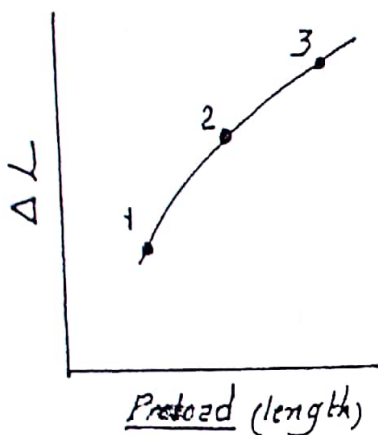
ΔL progressive

Load shortening curve

Preload

Afterload

⊖⊖



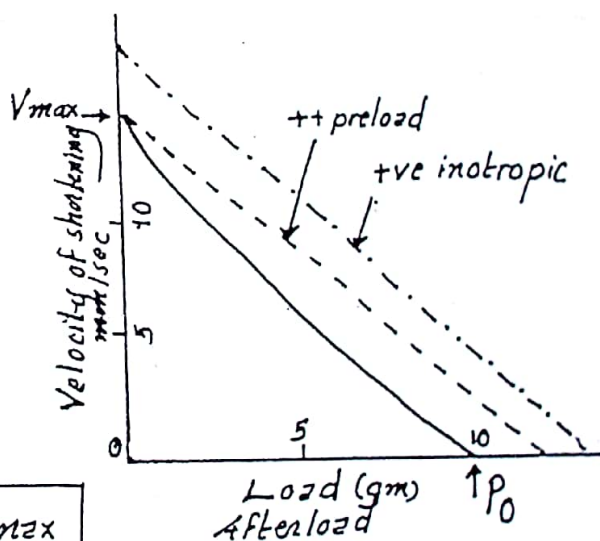
2 Length - velocity curve

Progressive ⊕⊕ in afterload

→ progressive ⊖⊖ in dl/dt

P_0 0 velocity at peak isometric tension

V_{max} Maximal velocity at 0 load (extrapolated point)



Effect of	Curve	P_0	V_{max}
1 ++ preload	shifts to Rt	++	constant
2 +ve inotropic	shift to Rt	++	⊕⊕

Inotropic state: Ability to contract independent of change in [preload, afterload]

CICR is below maximum i.e. ++ Ca^{++} → +ve & -- Ca^{++} → -ve inotropic

1 Length-tension curve +ve inotropic → ++ ΔL & vice versa

2 Load-velocity curve +ve inotropic → ++ dl/dt & vice versa

Frequency of contraction (force frequency relation) :

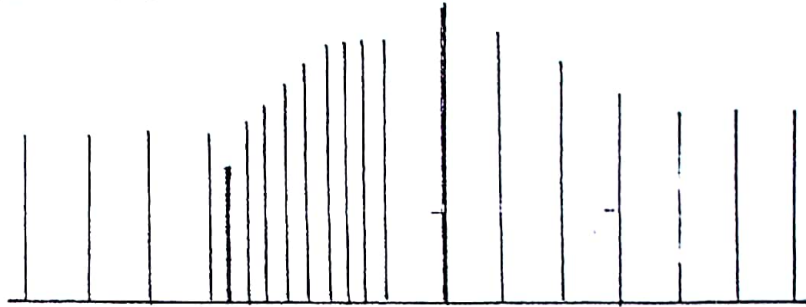
Stair case or treppe phenomenon

++ Frequency of stim. $\longrightarrow$ +ve inotropic state.
by changing Ca^{++} release & uptake

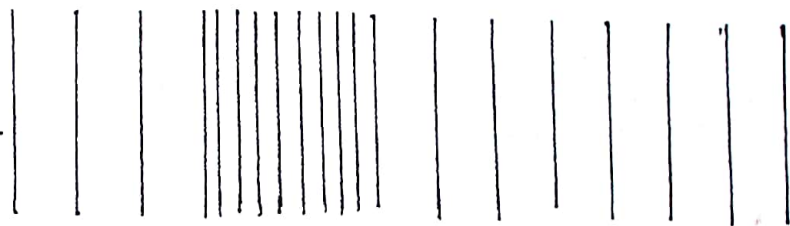
1st initial slight drop.

then progressive rise in force of contraction & vice versa

Contraction



Action potentials



In the whole heart :

- Length of m. fibres $\propto$ end diastolic volume.
- Tension $\propto$ pressure developed in ventricle
- Developed tension $\propto$ diastolic vol. (within limit)

Starling curve

- Preload = End diastolic volume
- Afterload = End systolic pressure = Aortic pressure
- Frequency = Heart rate.

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ECG

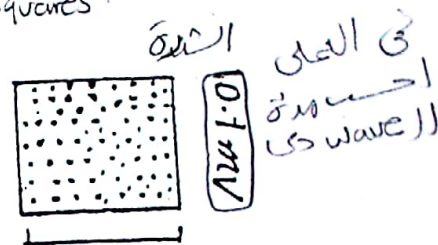
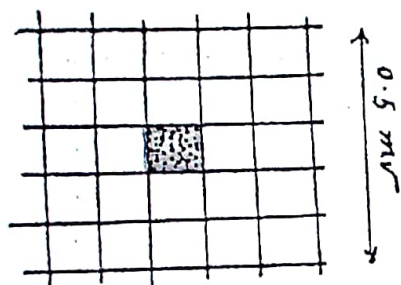
recorder of rapid voltage change of heart.
Biphasic action potential.

From the skin Body fluids are good conductor.

مراقبة

- Apparatus
 - 1 Monitoring machine ICU
 - 2 Recording machine fed to heat stylus pen recorder
- Calibrated strip of paper

P wave to Sd
2 small squares



So $\frac{0.04 \text{ sec.}}{40 \text{ msec.}}$

لا ريب
محررات

25 small square / second
1500 " " / minute

Vector : (Sum of AP)

Atrium

Septum



Daumeyer & Lt

Voltage



Upward & Rt

ventricle > atrium
mass of tissue



Downward's R



upwards & it

Duration $\propto \frac{1}{\text{velocity of conduction}}$

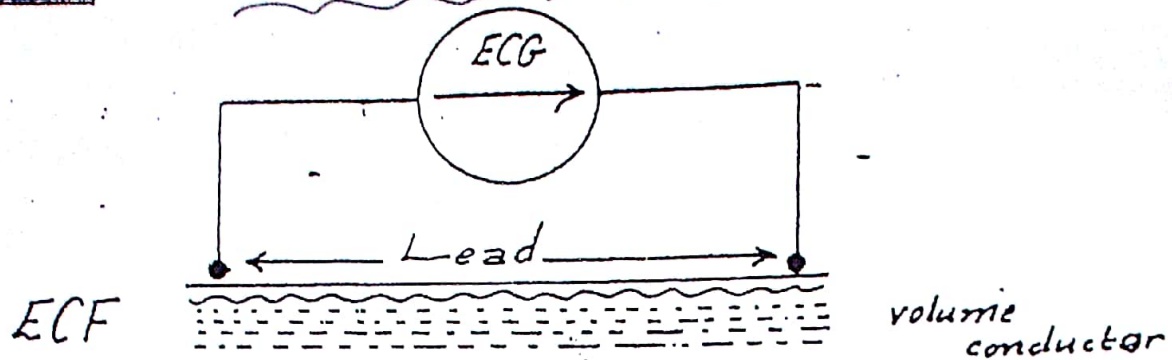
Polzrites

Depol is directed towards +ve electrode
positive wave (upward) & vice versa

Repol is directed towards +ve electrode
negative wave (downward) & vice versa

Vector is parallel to lead → maximal effect
 perpendicular to lead → not recorded.

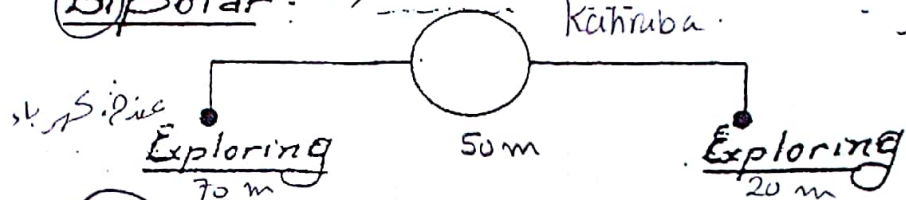
Lead : Position of the two electrodes



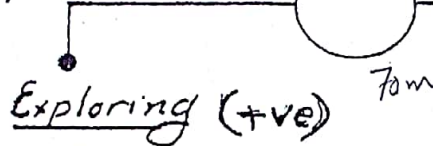
Types of leads :-

(A) According to TYPE of electrodes :

(1) Bipolar → 2 electrodes Zandoham Kahruba



(2) Unipolar (V) :



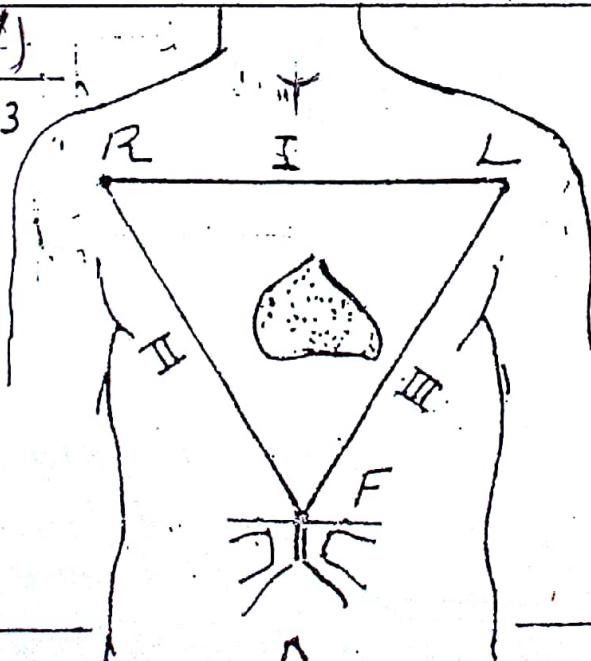
(B) According to SITE of electrodes :

(1) Limb Recording in FRONTAL plane

(2) Chest Recording in HORIZONTAL plane

Unipolar leads (V)

- Augmented limb 3
 aVR
 aVL
 aVF
- Chest 6 V₆
 V₁ to V₆
- Esophageal rarely used



Bipolar leads

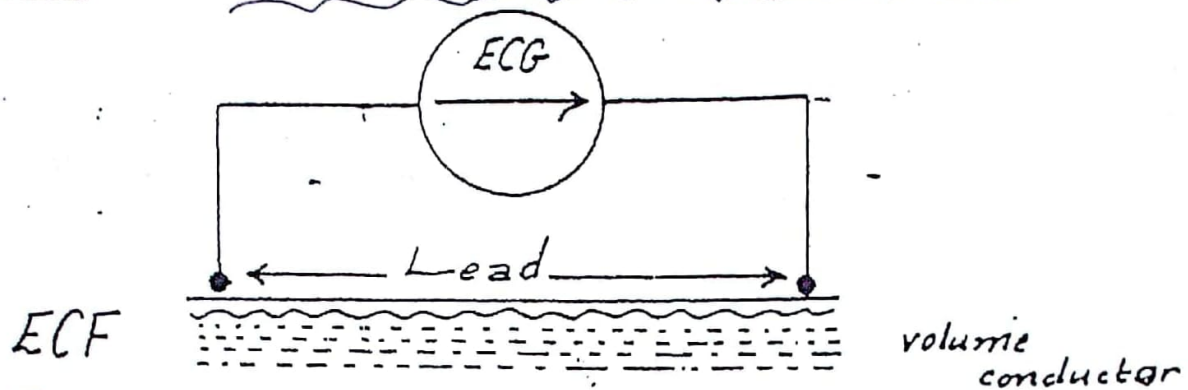
- Limb 3
 I
 II
 III

Cir 8

12/08/25

Vector is parallel to lead → maximal effect
 perpendicular to lead → not recorded.

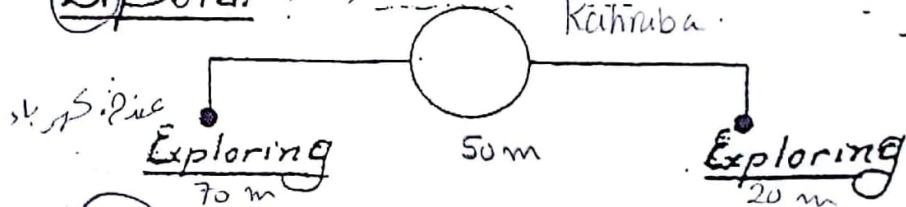
Lead : Position of the two electrodes



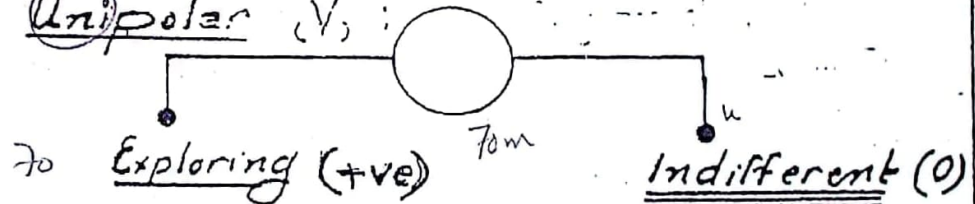
Types of leads :-

(A) According to TYPE of electrodes :

(1) Bipolar → 2 electrodes 3 and 40m Kaitruba



(2) Unipolar (V):



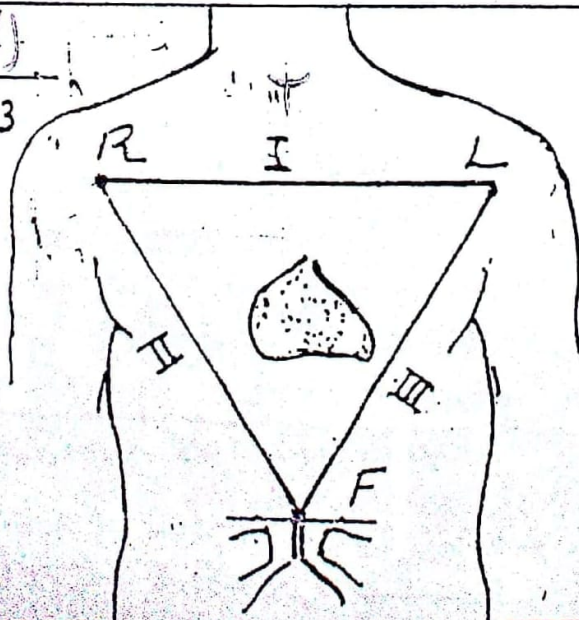
(B) According to SITE of electrodes :

(1) Limb Recording in FRONTAL plane

(2) Chest Recording in HORIZONTAL plane

Unipolar leads (V)

- Augmented limb 3
 aVR
 aVL
 aVF
- Chest 6 V6
 V1 to V6
- Esophageal rarely used



Bipolar leads

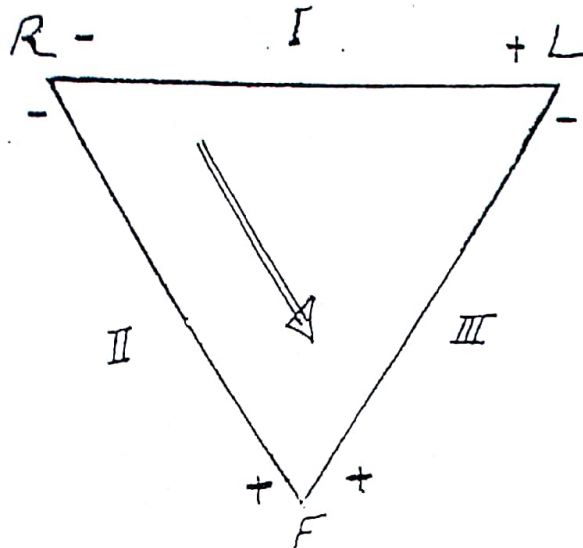
- Limb 3
 I
 II
 III

Cir

Bipolar limb leads

3

i.e. Standard limb leads



R = Rt arm

L = Lt arm

F = Lt foot

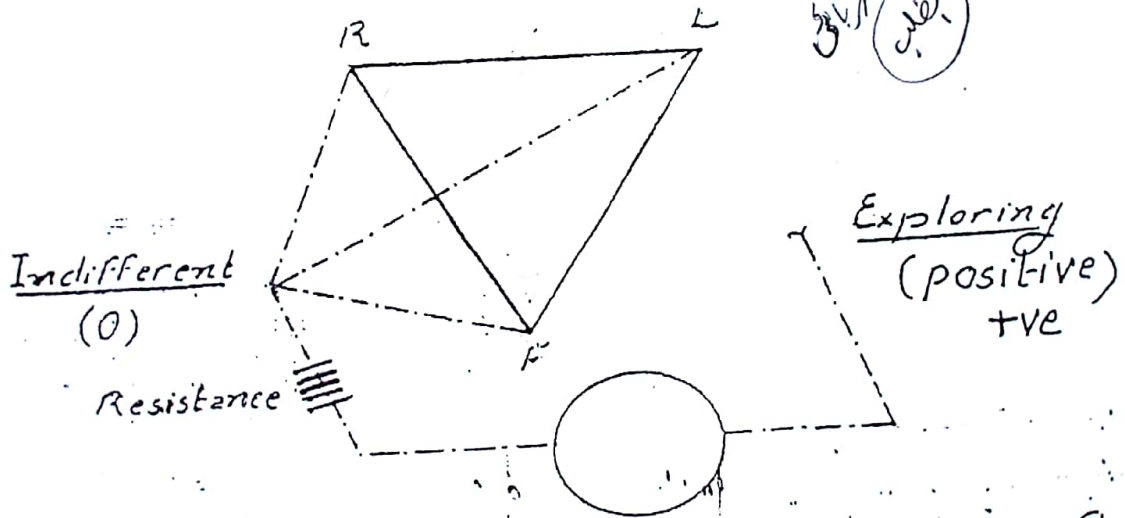
note

Rt foot earthing

Unipolar lead

9

V



$$\text{Indifferent} = (R - L) + (L - F) + (F - R) = 0$$

Unipolar limb leads

Exploring ——— Limb

Unipolar chest leads

Chest

Unipolar esophageal leads

Esophagus

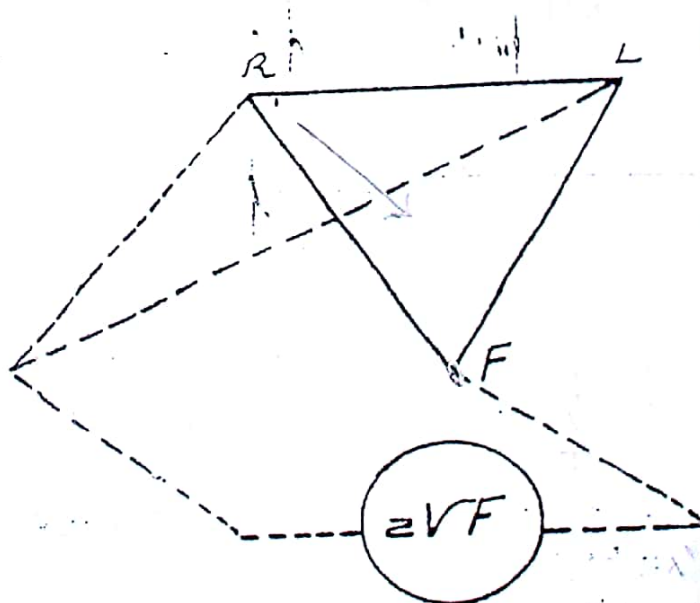
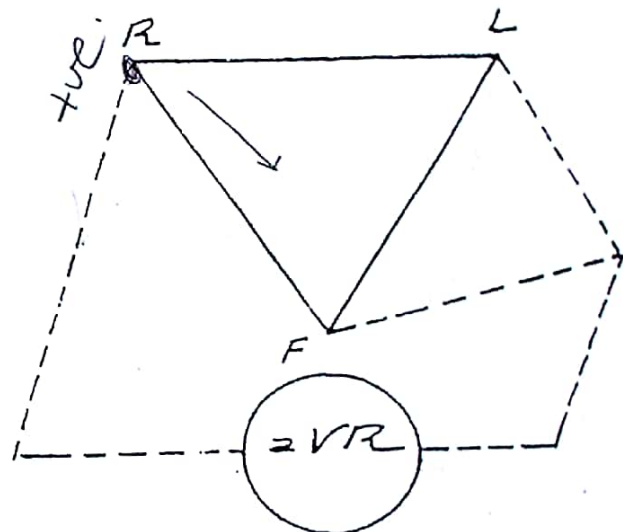
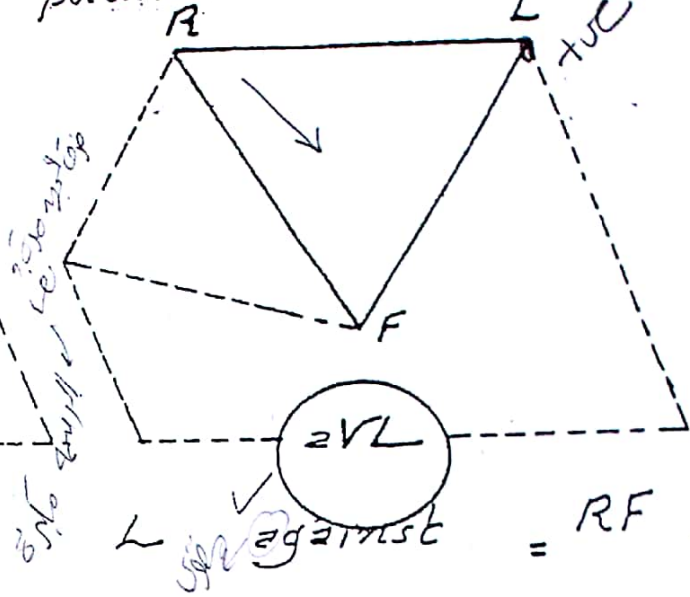
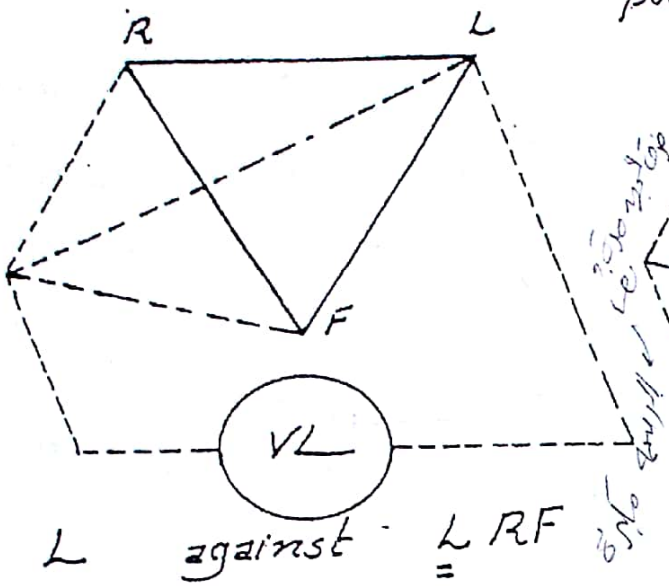
rarely used

Cir

Unipolar limb leads

Augmented unipolar limb leads

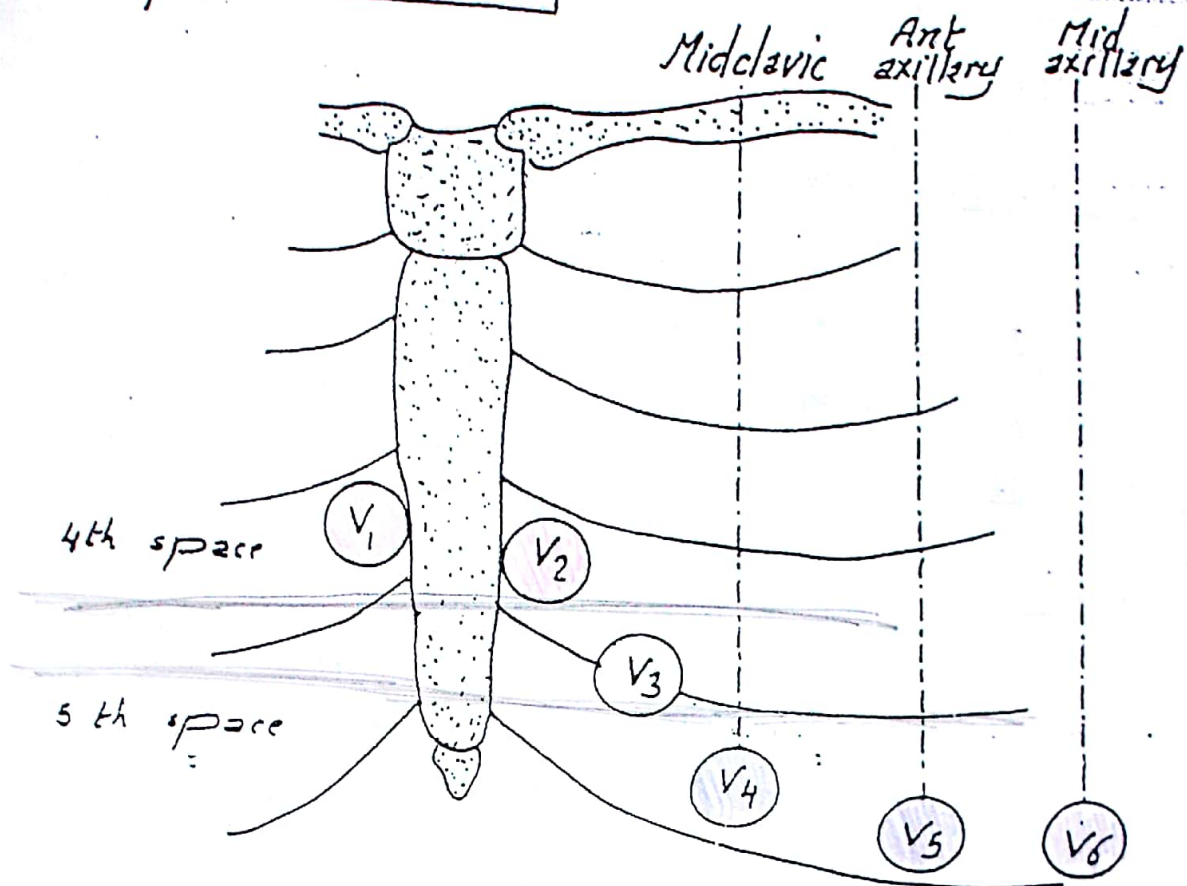
same configuration
potential is 50% more.



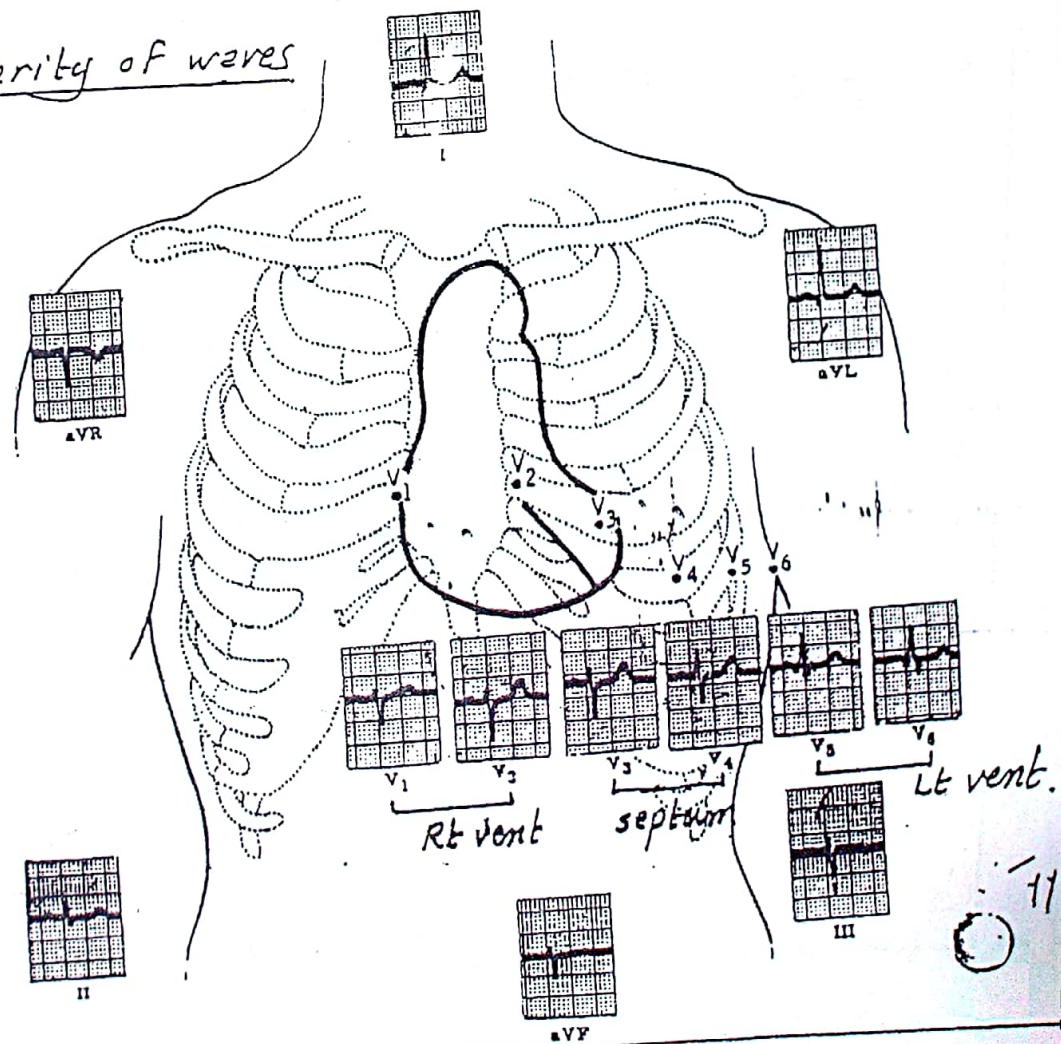
PIV
Exploring
50% more

012108/25
Ci

Unipolar chest leads

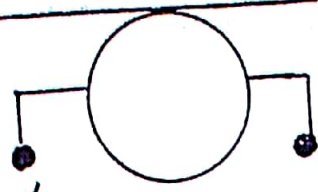


Polarity of waves



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Leads



Bipolar limb leads

	<u>Exp</u>	<u>Exp</u>
I	R	L +ve
II	R	F +ve
III	L	F +ve

small circle - L₁
 alternate lead
 electrode
 +ve

Augmented unipolar limb leads

	<u>Indiff</u>	<u>Exp</u>
aVL	RF	+ve
aVR	LF	L
aVF	RL	R

بالسلك
 To left and
 downward
 Right to left
 11 to left 11 to right
 +ve

Unipolar chest leads

	<u>Indiff</u>	<u>Exp</u>
V ₁	RLF	RL
V ₂	RLF	t
V ₃	RLF	t
V ₄	RLF	t
V ₅	RLF	t
V ₆	RLF	t

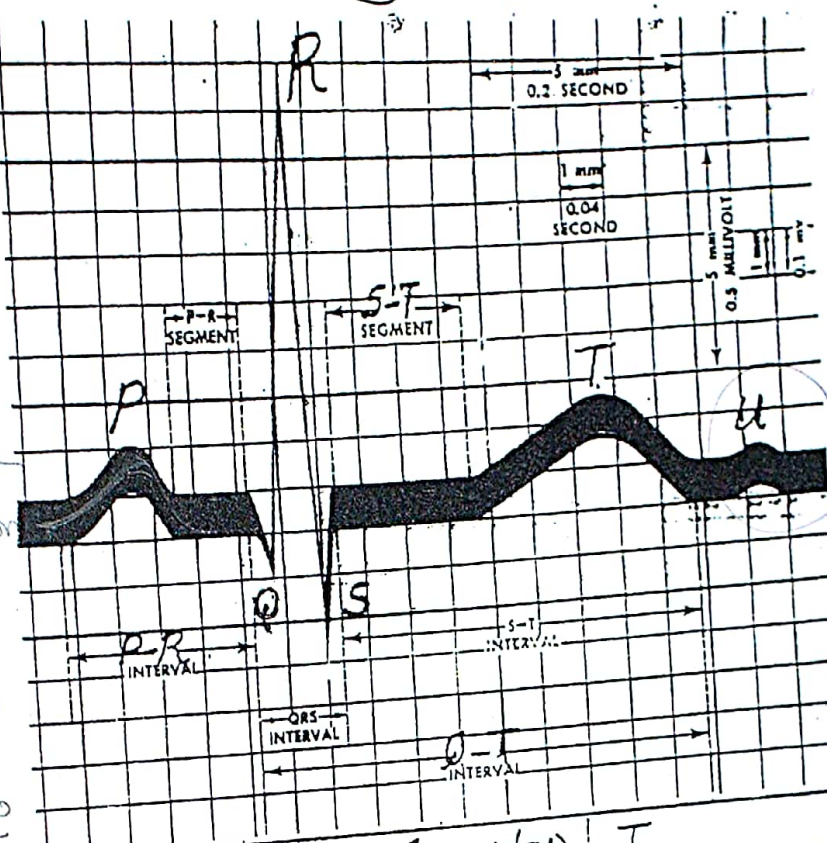
4th space parasternal
 4th space parasternal
 Between V₂ & V₄
 5th space mid clavicular
 5th space ant. axillary
 5th space mid axillary

Normal ECG

علاج

Waves + Segments = Intervals

from recording by one galvanometer 5 cm/sec
vent dep
atrial rep



* P = atrial depolarization (atrial dep) atrial systole
Da msh. Mechanical
AP

12) 100
Papillary muscle repolarization

WAVES	P	QRS (complex)	T	u
<u>Represents</u>	A. depol.	V. depol	V. repol.	Papillary m repo
<u>Duration</u>	0.08 - 0.15 i.e 80-100 msec	0.08 second speed bec. velocity & ↑ conduction سرعة	0.16 sec.	0.05 sec.
<u>Voltage</u> (height) limb leads chest leads	0.25 mV	1 mV 3-4 mV V1 V2 small R (shades) V5 V6 large R small S	> 0.5 mV > 1 mV	usually not recorded.
<u>Shape</u>	1st half for RT atrium 2nd half for LT atrium	R always +ve Q -ve before R S -ve after R	slightly rounded asymmetrical	
<u>Direction</u>	Inverted in aVR		Inverted in aVR	

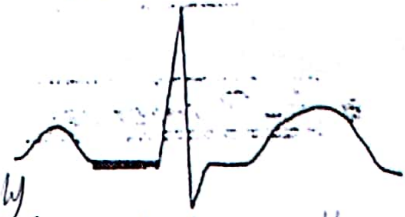
A repol: masked by QRS complex & very low voltage
SAN, AVN, AVB & Purkinje fibres: not recorded (small mass of tissue)
Q wave < 1/4 R if more than 1/4 R = myocardial infarction
appears in Lead I, aVL & aVF, V5 & V6
T wave (repol) in the same direction of R wave (depol.)
Last part to be depol (epicardium) is the 1st to be repol.

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Segments

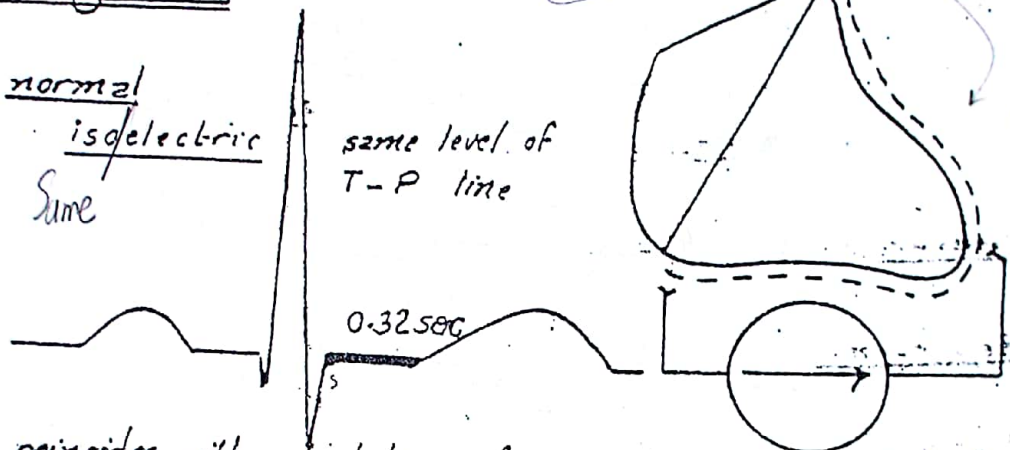
PR segment

- Measurement end of P to beginning of QRS _{usually}
- Represents conduction in AV node and AV bundle
- Normal 0.08 to 0.1 second



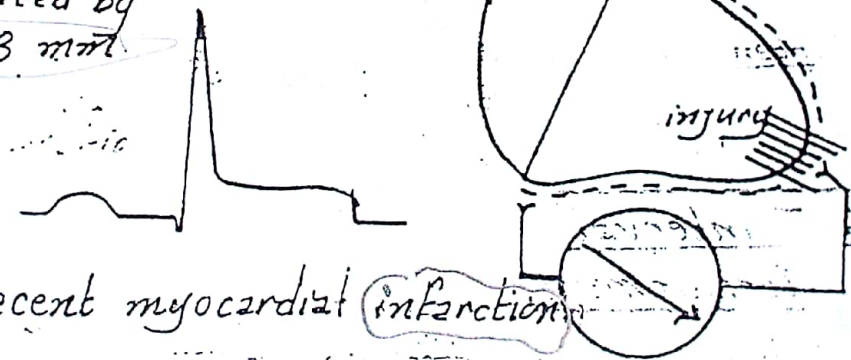
ST segment

- normal isoelectric _{same}
- coincides with plateau of monophasic action potential. i.e. 0.32 sec. i.e. vent repol.
- Injury elevated by 2-3 mm



- coincides with plateau of monophasic action potential. i.e. 0.32 sec. i.e. vent repol.

- Injury elevated by 2-3 mm



= Recent myocardial infarction

infarction

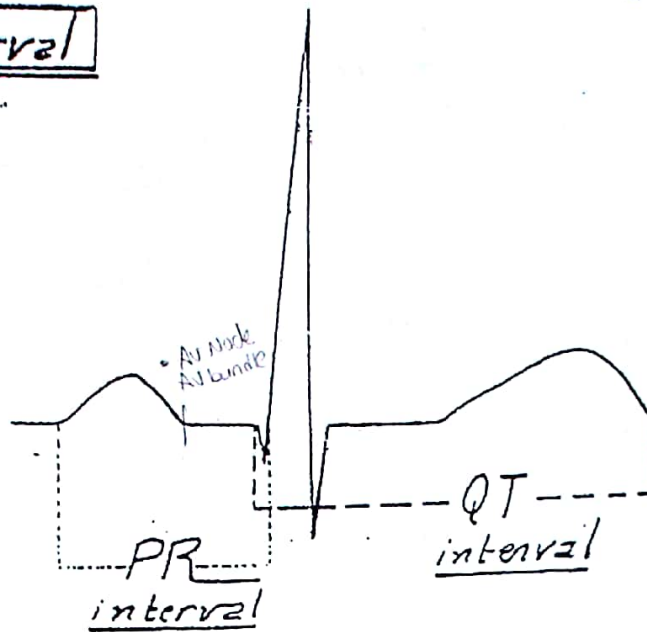
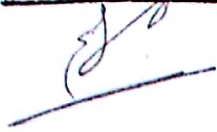


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Intervals

waves + segments

P-R interval



Measurement

Beginning of P
Beginning of R

Represents

A. depolarization
Conduction in AVN & AVB

Normal

0.16 - 0.20 sec (average 0.189 msec)

Prolonged

A-V Block
Atrial enlargement.

Shortened

Vagus stimulation (stimulation).
A-V nodal rhythm.
Symp. stimulation.

Q-T interval

Measurement

beginning of QRS complex
end of T

Represents

Ventricular depol + vent repolariz.

Normal duration

0.40 second (دقة المدة)

Prolonged

Hypocalcaemia

15'
12/08/25

200 mm → 25 mm
 200
 2 = 100

Abnormal
 Disorder

Comments on normal ECG:

1500 Per minute

1 Heart rate HR number of □ between 2 following R

normal 60 - 90 average 75/min.

2 Rhythm almost regular

SAN 66
 AVN 12

P is inverted

i.e R waves at regular intervals

3 Voltage i.e voltage of QRS in limb leads

normal > 1.6 mV i.e 16 mm.

4 Axis of the heart i.e vector of QRS in unipolar limb leads I, II & III (frontal plane).

Normal -30 to +90°



RT axis deviation

QRS in
 L1 -ve
 LIII +ve

180

+90° to -120°

Long slender person

Rt vent. hypertrophy
 Rt BBB
 Lt vent. infarction

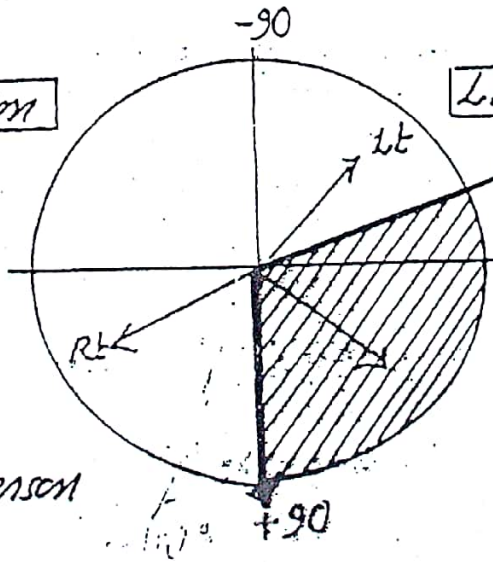
LT axis deviation

QRS in
 L1 +ve
 LIII -ve

-30° to -90°

Short stunted person.
 Full term pregnancy

Lt vent. hypertrophy
 Lt BBB
 Rt vent. infarction

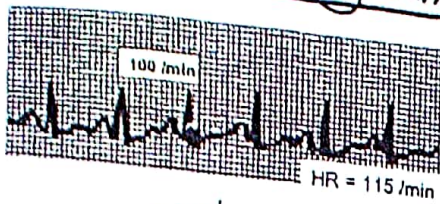


2012/08/25

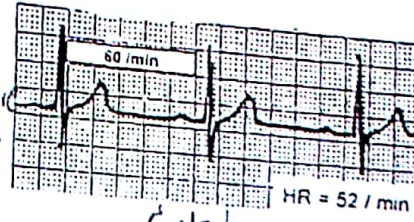
Abnormal ECG

A) Disorders of rhythm

عند نقطة ضربات القلب (معدل ضربات القلب)
 ① SAN ينشأ القلب
 Range 60 - 100/min
 80-100/min

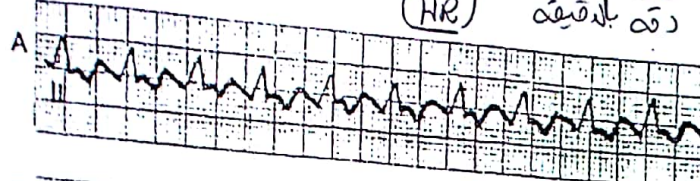


Per min
 1500
 عدد ضربات بين
 كل دقة 100

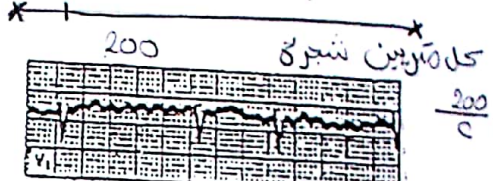


sinus tachycardia > 100/min
 (HR) دقة 150

sinus bradycardia < 60 min.

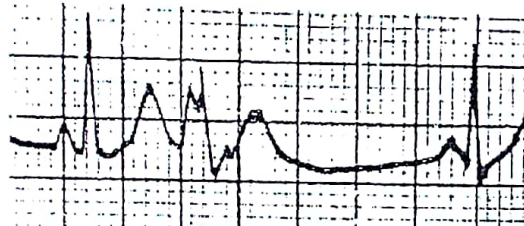


Atrial Flutter A 2:1 B 4:1



A. Fibrillation

NO

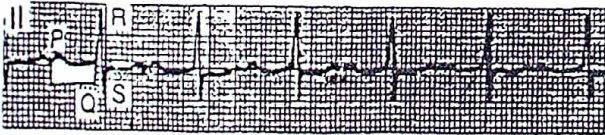


Ventricular extrasystole (PVBs)

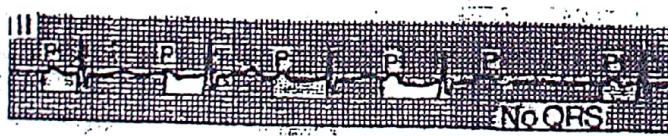
SAN
 AVN
 AVN
 atrial

B) Disorders of conduction

A-V block i.e Heart block

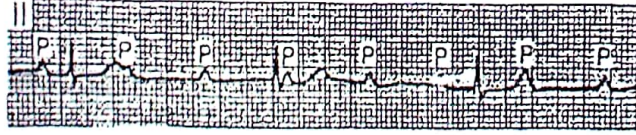


1st degree



2nd degree

Wenckebach



3rd degree

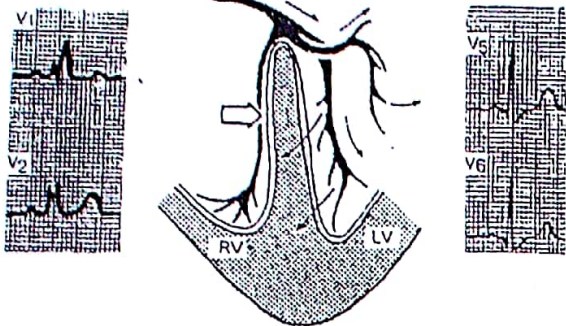
Complete heart block

Dep
 atrial
 ventricle
 في نفس الوقت

Both Ventricle
 AVN 20 PR interval

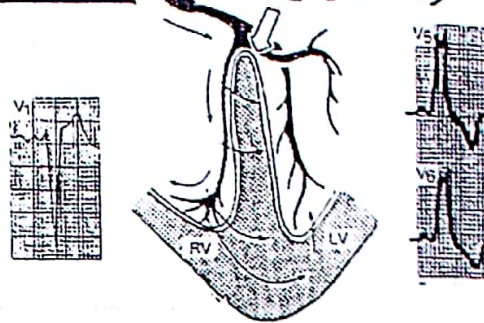
Bundle branch block BBB

Rt BBB (V_1 & V_2)



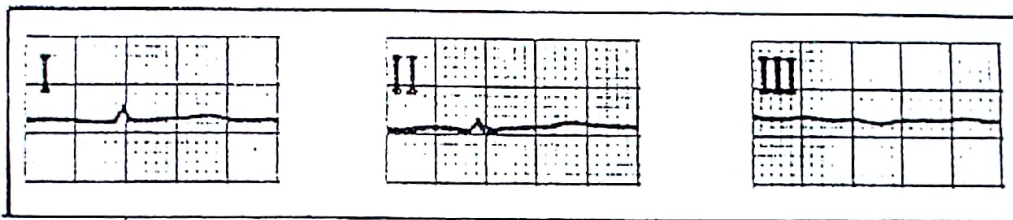
Delayed RV activation
Double hump QRS

Lt BBB (V_5 & V_6)



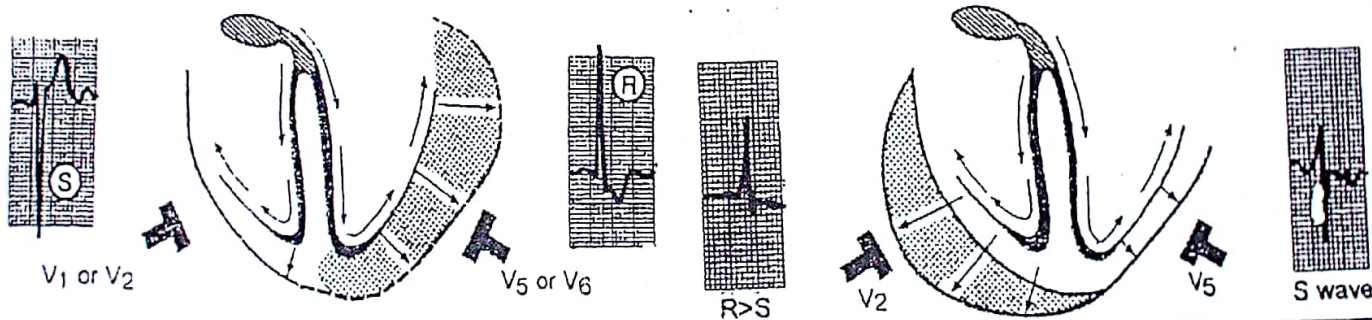
Delayed LV activation
Double-hump QRS

C) Low voltage ECG e.g. hypothyroidism (myxedema)

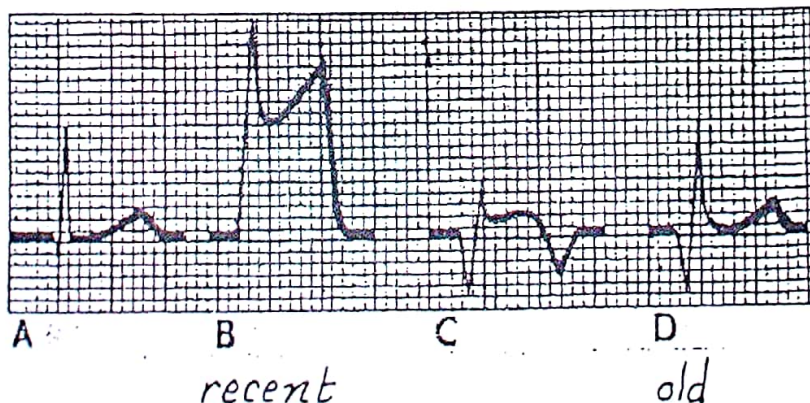


D) Lt ventricular hypertrophy

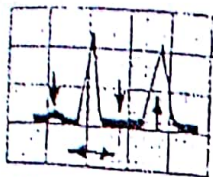
Rt ventricular hypertrophy



E) Myocardial infarction



F) Electrolyte imbalance



Hyperkalemia



Hypokalemia

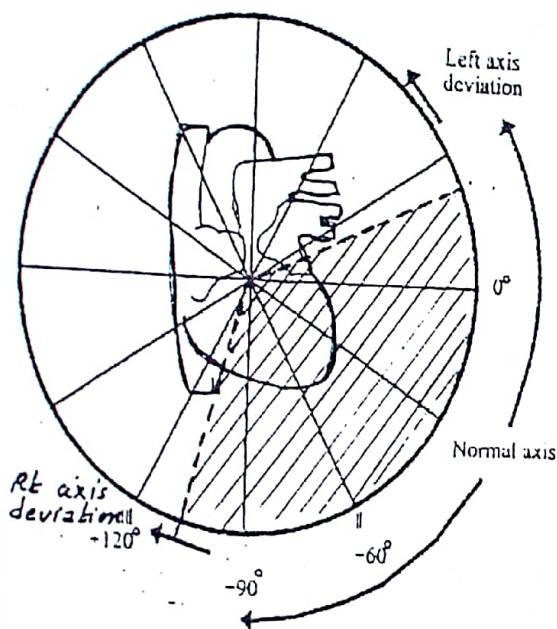


Hypercalcemia



Hypocalcemia

G) Axis deviation



	L_1	L_{II}	L_{III}
Normal			
Lt axis deviation			
Rt axis deviation			

Cardiac cycle

8 phases

8 changes

1 Atrial systole

- 2 Isovolumetric contraction
- 3 Maximal (rapid) ejection
- 4 Reduced ejection

5 Protodiastolic

- 6 Isovolumetric relaxation
- 7 Maximal Filling
- 8 Reduced Filling

Ventricular Systole 0.3 second

2, 3 & 4

Ventricular Diastole 0.5 second

Early

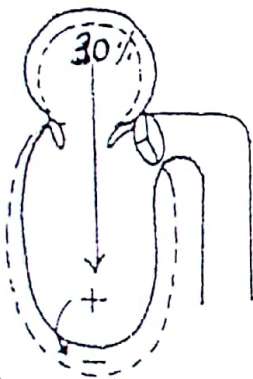
5, 6 & 7

Mid

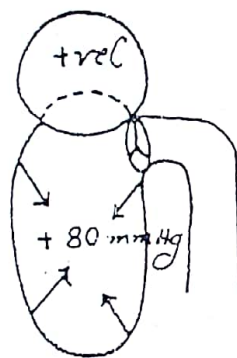
8

Late

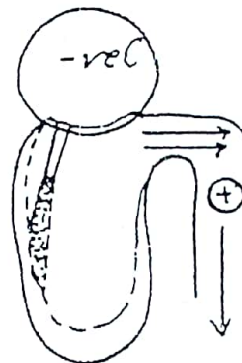
1



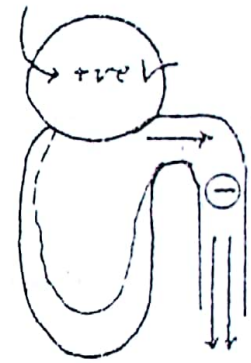
A. systole



Isovolumetric cont.



Maximal ejection



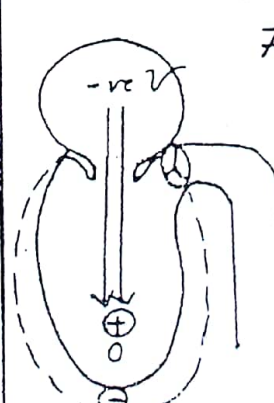
Reduced ejection



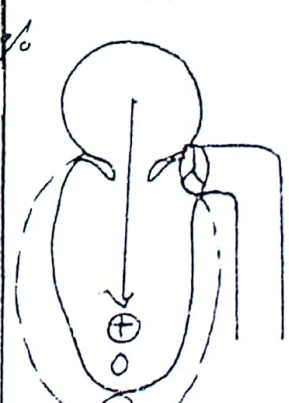
Protodiastolic



Isovolumetric relax.



Maximal filling

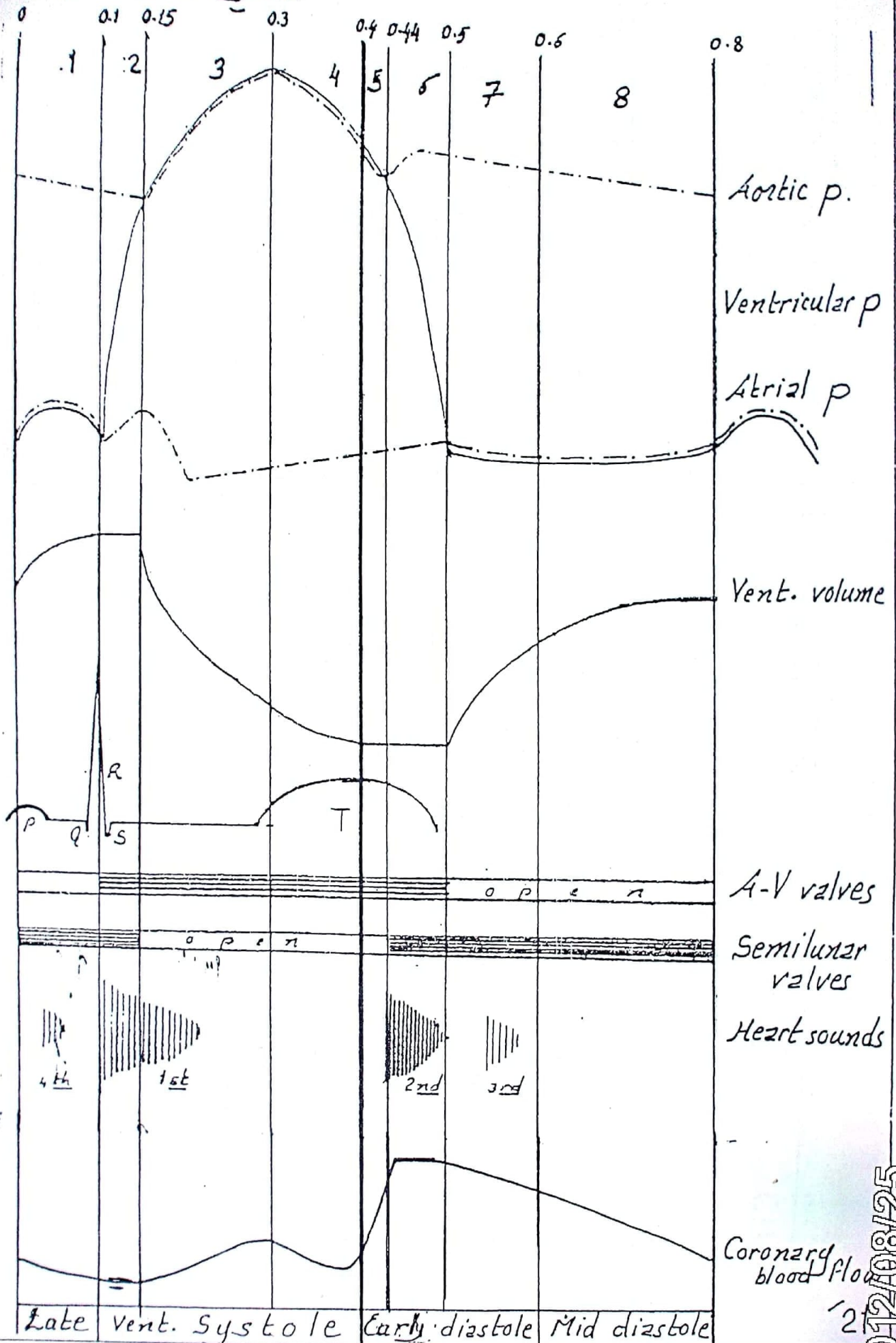


Reduced filling

20

12/08/25

Cardiac cycle



Definitions

Stroke volume
SV

$$SV = EDV - ESV$$

$$130 - 60$$

Minute volume
Cardiac output
COP

Minute

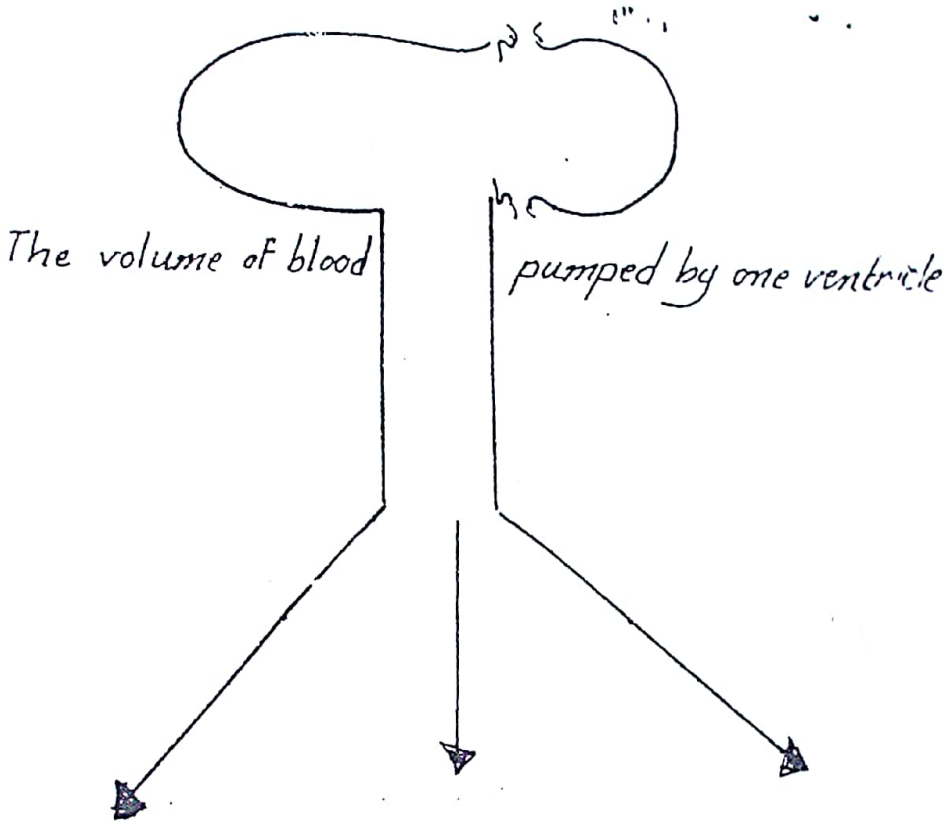
$$COP = HR \times SV$$

$$72 \times 70$$

Cardiac index
CI

$$5/1.7 = 2.94 \text{ L/m}^2/\text{min}$$

Cardiac output



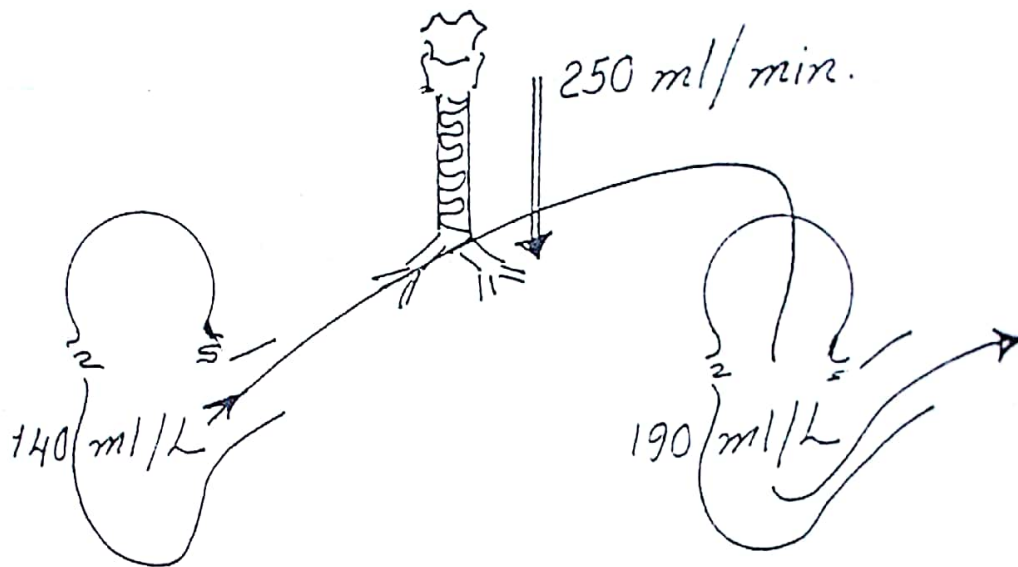
EDV = End diastolic vol. ESV = End systolic vol. HR = Heart rate.

Cir 22

Direct Fick method

$$F = \frac{\text{Amount of substance taken / min}}{\text{Arterio-venous difference}}$$

$$\begin{aligned} \text{COP} &= \frac{\text{O}_2 \text{ consumption (ml/min)}}{\text{Arterio-venous O}_2 \text{ difference}} \\ &= \frac{250 \text{ ml/min}}{190 \text{ ml/L} - 140 \text{ ml/L}} \end{aligned}$$



Conditions that affect COP

- ± Sleep moderate temp changes
- + Excitement upto 100%
- Exercise 70%
- Eating 30%
- Epinephrine
- Hot atmosphere
- Pregnancy
- Lying to standing
- Rapid arrhythmia
- Heart diseases

Control of COP

- 1 VR primarily determinant
- 2 Cardiac performance

Under normal conditions, $COP = VR$ within limit of Cardiac performance

Factors affecting VR

Ohm's law

$$F = \frac{\Delta P}{R}$$

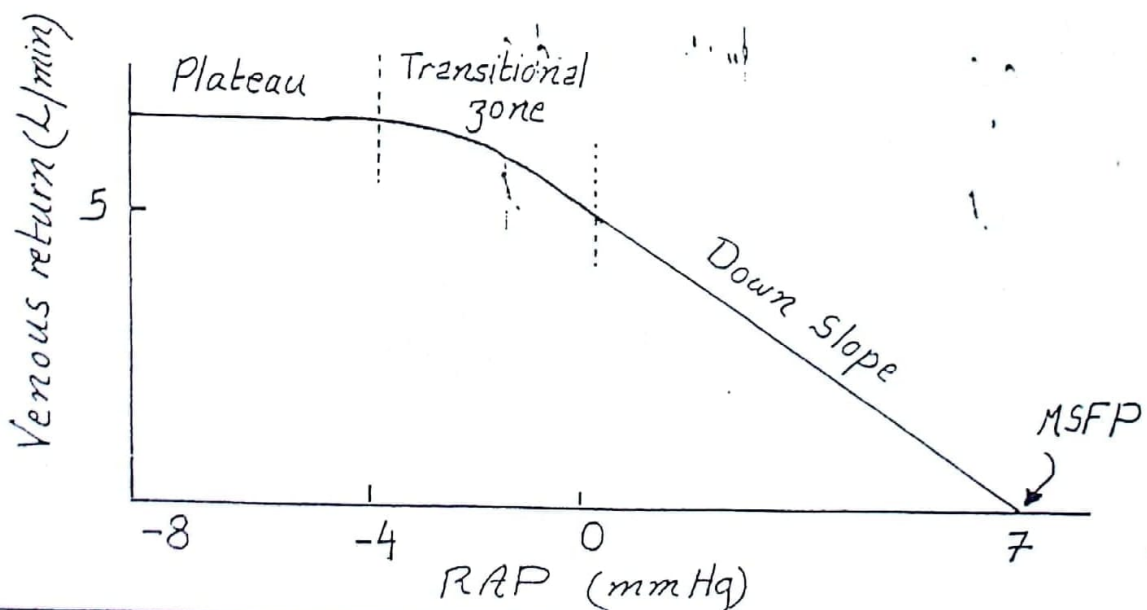


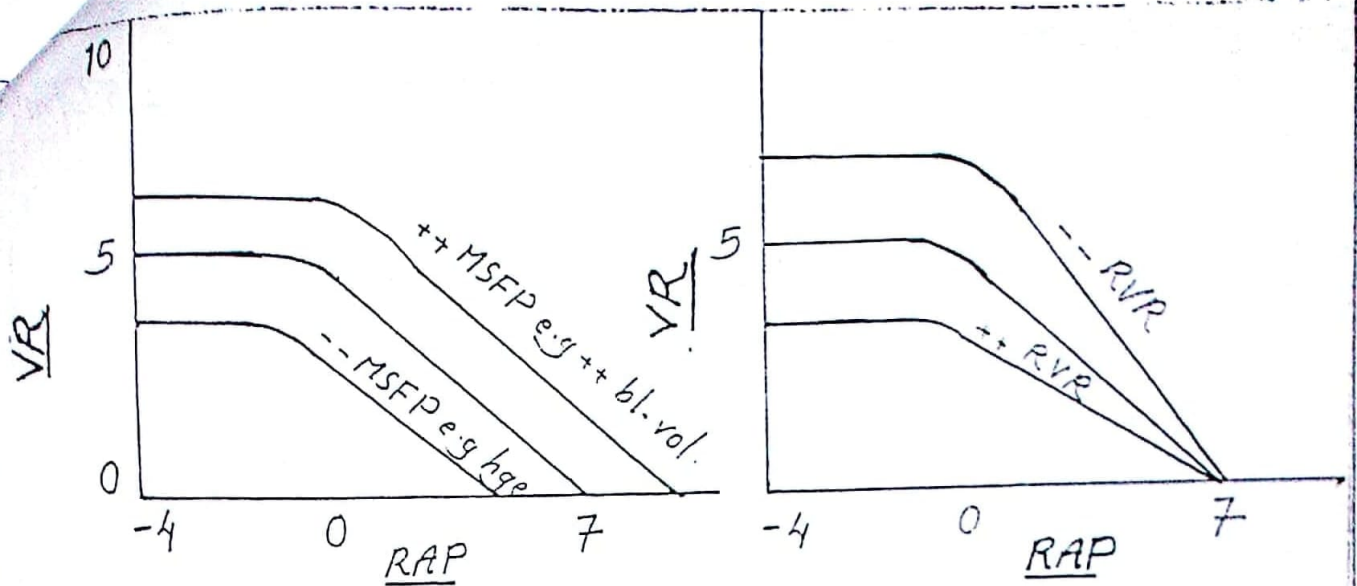
$$VR = \frac{MSFP - RAP}{RVR}$$

1 MSFP 6-8 mmHg $\propto \frac{\text{Volume of blood}}{\text{Capacity of vascular system}}$

2 RAP (CVP) 0-2 mmHg $VR \propto \frac{1}{RAP}$

Venous return (Venous Function) curves:





3 RVR

$$F = \frac{\Delta P}{R}$$

$$R = \frac{R_{\Delta P}}{F} = \frac{MSFP - RAP}{VR} = \frac{7 - 0}{5} = 1.4 \text{ mmHg/L/min}$$

Cardiac output curve

$COR \propto RAP$ within limits

Explanation: ++ RAP $\rightarrow$ ++ C. Filling $\rightarrow$ ++ EDV (preload)
 $\rightarrow$ ++ C contractility Starling law $\rightarrow$ ++ COP

